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Quality Management in Healthcare - The Foundation of Patient Safety

Quality management and patient safety are the two pillars upon which every successful healthcare institution is built. Quality is not merely about providing treatment-it is about providing the **right treatment, to the right patient, at the right time, through the right procedure, with the highest standards of safety.** If quality is compromised, patient safety is inevitably at risk.

In medical practice, even a single mistake can have devastating consequences. Administering the wrong medication, giving the correct drug through the wrong route, operating on the wrong patient, or performing surgery on the wrong side are errors that can change lives forever. Unlike many other professions, healthcare does not always offer a second chance. A patient who loses confidence because of a preventable error may never return, and more importantly, the consequences may be irreversible.

Quality assessment has become an essential part of every industry. Whether it is the manufacture of automobiles, mobile phones, aircraft, jewellery, or consumer products, stringent quality checks are carried out before a product reaches the customer. If industries producing machines and products demand uncompromising quality, healthcare-which deals with human life-must uphold even higher standards.

One of the most widely accepted benchmarks of quality is **Six Sigma,** a methodology that strives for near perfection by limiting defects to only **3.4 defects per million opportunities.** This remarkable level of precision demonstrates what can be achieved through systematic quality management, continuous monitoring, standardization, and ongoing improvement.

At KG Hospital, quality is not an occasional exercise but a way of life. Comprehensive quality systems are implemented across all **75 departments,** ensuring that every patient receives safe, ethical, evidence-based, and compassionate care. Standard operating procedures, clinical protocols, regular audits, continuous training, and performance monitoring have become part of our organizational culture.

Quality is not only a professional responsibility but also a driver of institutional growth. When patients recognize that a hospital consistently provides safe, reliable, and compassionate care, they place their trust in that institution. Trust leads to confidence, confidence leads to reputation, and reputation leads to sustained growth.

The journey of KG Hospital stands as testimony to this philosophy. From a modest **10-bed hospital,** KG Hospital has grown into a **250-bed tertiary care institution** with **75 departments** and a dedicated workforce of **more than 1,500 employees.** What once served primarily the local community has today become an internationally recognized healthcare organization, attracting patients from across India and abroad.

This unwavering commitment to quality, patient care, education, research, and social responsibility has earned KG Hospital and its Chairman numerous prestigious recognitions. These include the **Dr. B. C. Roy National Award,** the **Padma Shri,** and several awards acknowledging excellence in healthcare and humanitarian service, particularly the institution's commitment to providing quality treatment for those who cannot afford it. Among thousands of healthcare institutions in the country, Dr. G. Bakthavathsalam has also been recognized by professional healthcare organizations as a distinguished healthcare **luminary.**



Padma Shri. **Dr. G. Bakthavathsalam**
Chairman, KG Hospital

What is a luminary ?

A luminary is an individual whose vision, leadership, knowledge, and lifelong contributions illuminate the path for others. Such recognition is reserved for those whose work transforms institutions and inspires generations of healthcare professionals.

Quality is not the responsibility of one department alone. It is the responsibility of every doctor, nurse, technician, administrator, and support staff member. It begins the moment a patient enters the hospital and continues until the patient safely returns home. Every interaction, every diagnosis, every procedure, and every decision must reflect our commitment to excellence.

At KG Hospital, our philosophy is simple:

Quality ensures safety. Safety builds trust. Trust creates excellence.

As we continue our journey of service, let us reaffirm our commitment to maintaining the highest standards of quality in everything we do. Every patient deserves nothing less.

Long live India.

Long live KG Hospital.

May every patient who comes to KG Hospital receive safe, compassionate, and quality healthcare and return home with renewed health and hope.

CAUTI-Associated Hyperammonaemia with Concurrent Stenotrophomonas maltophilia Bacteraemia: A Novel Hypothesis for Non-Hepatic Ammonia Dysregulation



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Introduction

Hyperammonaemia - serum ammonia exceeding 35 mcg/dL - is a metabolic emergency with potential for cerebral oedema, encephalopathy, coma, and death. The textbook association with hepatic failure has created a dangerous clinical blind spot: clinicians frequently fail to consider non-hepatic causes in patients with intact liver function.

Non-hepatic hyperammonaemia accounts for fewer than 5% of cases in the critically ill, yet its consequences are equally devastating when missed. Infection due to urease-producing bacteria represents the most clinically under recognised infectious route: bacteria hydrolyse bladder urea to ammonia, which is systemically absorbed - overwhelming the hepatic urea cycle even in individuals without underlying liver disease.

The Case

A 58-year-old agriculturalist woman, known hypertensive and type 2 diabetic, and recently post-left total knee replacement (TKR), presented with acute-onset altered sensorium, slurred speech, and tremors. There was no history of alcohol use, hepatic illness, nausea, seizures, or focal neurological deficit. She had been on native (herbal/Ayurvedic) medications for approximately two years.

On examination, she was conscious but disoriented (time, place, person), with impaired immediate memory, slurred speech, bilateral extensor plantar responses, bilateral knee and ankle clonus, and intention tremor. Crucially, there were no history of chronic liver disease - no spider naevi, palmar erythema, caput medusae, or organomegaly. Resting tachycardia and patchy bilateral basal crepitationswere noted.

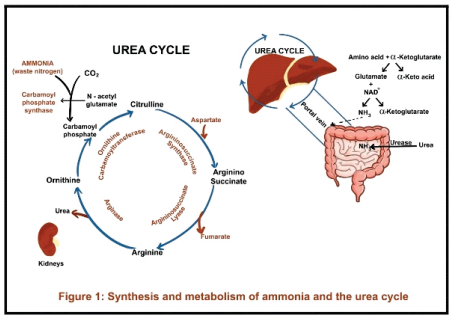


Figure 1: Synthesis and metabolism of ammonia and the urea cycle

Investigation	Result
Serum Ammonia	131.8 mcg/dL (Ref: 11 – 35)
AST / ALT	10 / 14 U/L
Total Bilirubin	0.23 mg/dL
Albumin / Total Protein	3.0 / 5.6 g/dL
Fibroscan	Normal
MRI Brain	Normal parenchyma
Urine Culture	ESBL E. coli (CAUTI confirmed)
Blood Panel PCR	Stenotrophomonas maltophilia
EEG / CT Brain	Normal / chronic lacunar infarct

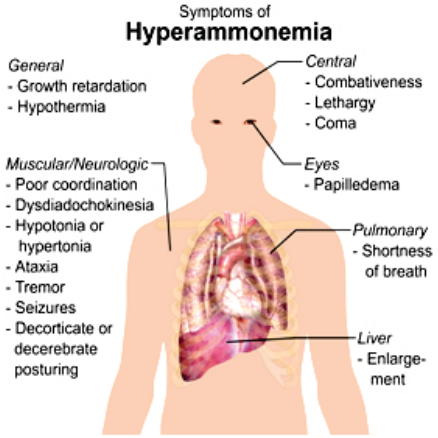


Table 1. Key investigations with clinicopathological interpretation.

The clinical picture was thus one of **acute hyperammonaemia encephalopathy** which on further evaluation has confirmed CAUTI (ESBL E. coli), Stenotrophomonas maltophilia bacteraemia(blood PCR), and complete **absence of any hepatic, structural, metabolic, or toxicological aetiology.** This directed diagnostic focus firmly toward an infectious-metabolic mechanism.

NON-HEPATIC HYPERAMMONAEMIA: A FRAMEWORK FOR CLINICIANS

When faced with hyperammonaemia and intact liver function, the following aetiological categories should be systematically considered:

Category	Mechanism	Examples
Infectious (UTI/CAUTI)	Urease hydrolyses bladder (urea ? 2NH3); systemic absorption	Proteus, Klebsiella, Pseudomonas, Ureaplasma, Staphylococcus.
Bacteraemia/Sepsis	Systemic proteolysis, catabolism; overwhelmed urea cycle	GNB sepsis; S. maltophilia (proposed)
Portosystemic shunting	Ammonia bypasses hepatic detoxification	TIPS, congenital shunts
Drug-induced	Urea cycle inhibition; carnitine depletion	Valproate, ribavirin, L -asparaginase
Urea cycle defects (adult)	Partial enzyme deficiency unmasked by stress	OTC deficiency (most common in adults)
Malignancy-associated	Haematological: poorly understood nitrogen dysregulation	Myeloma, leukaemia, lymphoma
GI protein load / Small intestinal bacterial overgrowth(SIBO)	Bacterial urease in gut; excess substrate from bleed	Upper GI bleed, small bowel overgrowth
Parenteral nutrition	Excess amino acid load (especially glycine)	ICU patients on TPN

Table 2. Differential diagnoses of non-hepatic hyperammonaemia.

3.1 The CAUTI–Hyperammonaemia Pathway

The enzymatic mechanism is well-established: bacterial urease (EC 3.5.1.5) catalyses urea hydrolysis —

Urea - NH_3 + [H_2 NCOOH]- 2NH_3 + CO-

Normal urine contains 200–400 mmol/L urea — a rich substrate reservoir. In CAUTI, an inflamed urothelium and indwelling catheter create conditions for both **enhanced bacterial ureaseactivity** and increased ammonia absorption. Even with a competent liver, a sufficiently high ammonia load from continuous vesical production can exceed hepatic urea cycle capacity, leading to systemic hyperammonaemia.

3.2 Hyperammonaemia in Sepsis / Bacteraemia

Certain **organisms hydrolyse urea into ammonia** and carbon dioxide, generating a local and then systemic ammonia load. In the urinary tract, this hydrolysis **alkalinizes the urine,** which paradoxically **increases the proportion of ammonia** present as the lipid-soluble, electrically neutral NH- species (rather than the charged NH-) by as much as 50%, compared with only 5–10% at neutral pH. Because NH- is lipid-soluble it passively **diffuses into urothelial cells**. Once inside the cell, at near-neutral intracellular pH, NH is converted to the far less membrane-permeable NH, which becomes trapped — a phenomenon termed “diffusion trapping.” a The trapped ammonium then **enters the systemic circulation through the venous drainage of the bladder** (bypassing first-pass hepatic detoxification). Independent of any specific organism, sepsis itself induces a hypercatabolic state. **Accelerated skeletal muscle proteolysis** increases the systemic nitrogen/ammonia load while simultaneously suppressing the muscle's normal glutamine-synthetase-mediated buffering capacity, by which excess ammonia is ordinarily stored as glutamate/glutamine in muscle. This combined **surge in production and loss of peripheral buffering** can saturate the urea cycle's reserve capacity, or unmask a previously silent partial urea cycle enzyme deficiency (most often a heterozygous ornithine transcarbamylase, OTC, defect) that only becomes clinically apparent during a period of severe metabolic stress such as sepsis, the postpartum period, or major surgery.

3.3 Downstream Neurotoxicity — Final Common Pathway

Regardless of the inciting mechanism, ammonia produces neurological injury through a shared pathway. **Ammonia freely crosses the blood–brain barrier;**

astrocytes, the only brain cells expressing glutamine synthetase, convert it to glutamine. Excess glutamine accumulation causes **osmotic astrocyte swelling**, which can progress to cerebral edema. This is compounded by mitochondrial/tricarboxylic acid (TCA) cycle dysfunction, NMDA receptor–mediated excitotoxicity, and oxidative stress from reactive oxygen species, ultimately producing the **encephalopathy, seizures,** and — in severe cases — coma characteristic of a hyperammonaemia crisis [1,2].

4. STENOTROPHOMONAS MALTOPHILIA: FROM BACTERAEMIA TO AMMONIA — THE HYPOTHESIS



4.1 Microbiology at a Glance

Stenotrophomonas maltophilia (previously Xanthomonas maltophilia, Pseudomonas maltophilia) is a **non-fermentative, aerobic, Gram-negative bacillus** ubiquitous in hospital water systems and indwelling devices. It is oxidase-negative, catalase-positive, DNase-positive, and produces characteristic **yellow-green colonies**. It has emerged as the leading carbapenem-resistant Gram-negative cause of bloodstream infections in some US cohorts, with crude mortality from bacteraemia ranging 14–69%.

Mechanism	Evidence Base	Ammonia Relevance
Serine/Metallo-proteases (StmPr1, StmPr2, StmPr3)	Well-characterised virulence factors; confirmed in clinical strains (Bhaumik et al., Frontiers CIM 2024)	Degrade host proteins → release α-amino groups → deamination → NH3
Nitrogen metabolism genes	Genome of S. maltophilia P13 encodes ammonium assimilation, glutamate synthase, glutamate - ammonia-ligase (Sherpa et al., 2021)	Active nitrogen cycling with ammonium as core metabolite
Deaminase / amino acid catabolism	Active catabolism of glutamine, asparagine, serine yields NH 3 directly; organism utilises ammonia as nitrogen source in minimal media	Non-urease-dependent ammonia generation in protein -rich environment (blood)
Culture ammonia odour	Documented microbiological observation — pungent NH3 odour on blood agar/MacConkey; attributed to proteolytic/deamination activity	In vitro proof -of-concept for active ammonia production
Bacteraemia = systemic exposure	Bloodstream presence provides access to circulating proteins, hepatic substrate, and systemic amino acid pools — far beyond localised CAUTI	Greater substrate availability compared to urine; potential to overwhelm hepatic urea cycle

Key risk factors for S. maltophilia bacteraemia includes **indwelling vascular/urological devices, prior antibiotic exposure, prolonged hospitalisation, prior surgery, diabetes, and immunocompromise.** The organism thrives in biofilm on urinary catheters, central venous catheters, and endotracheal tubes.

4.2 Why S. maltophilia May Generate Ammonia in Vivo — The Mechanistic Argument

Several biochemical properties of S. maltophilia support a plausible role in systemic ammonia dysregulation, particularly in the bacteraemia state where the organism circulates in blood with access to systemic protein substrates:

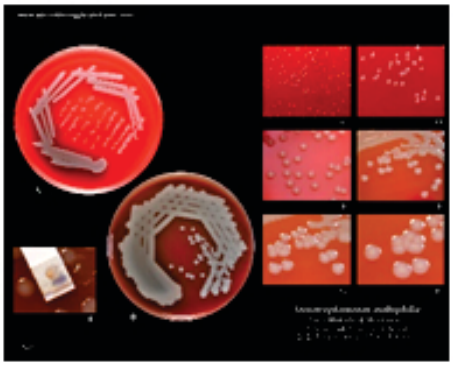


Table 3. Mechanistic pathways supporting S. maltophilia's potential role in systemic ammonia dysregulation.

4.3 The Bacteraemia Distinction — Why Blood Matters More Than urine

Isolation of S. maltophilia from blood — rather than urine — fundamentally alters the clinical significance. Bacteraemia implies **systemic dissemination with direct access to circulating nitrogen-containing substrates** (albumin, amino acids, acute-phase proteins), bypassing localised urothelial barriers. The bacteraemia state therefore offers a far greater substrate reservoir for ammonia generation than an isolated urinary isolate would, and carries a significantly higher systemic metabolic impact\

4.4 Scientific Caveats and Intellectual Honesty

The hypothesis that S. maltophilia bacteraemia directly causes clinically significant hyperammonaemia remains unproven. No human case series, controlled studies, or animal model data currently establish a causal link. **S.**

maltophilia shows variable urease results (often negative on standard biochemical panels), and its ammonia production in liquid media at 37°C has **not been formally quantified.** The possibility that bacteraemia represents a secondary consequence of the patient's critical state — rather than an **independent ammonia-generating driver — cannot be excluded.** Co-infection with ESBL E. coli as the dominant CAUTI pathogen remains the principal established mechanism in this case.

Nevertheless, this is the first documented case of concurrent S. maltophilia bacteraemia and CAUTI-associated non-hepatic hyperammonaemia, making it an important index observation that warrants formal investigation.

5. MANAGEMENT OF CAUTI-ASSOCIATED HYPERAMMONAEMIA

Management is directed along three concurrent axes: (a) treat the underlying infection, (b) reduce ammonia production/absorption, and (c) enhance ammonia elimination.

- **Catheter removal / replacement**
- **TMP-SMX (for S. maltophilia bacteraemia)**
- **Lactulose 15–30 mL TDS**
- **Rifaximin 550 mg BD**
- **L-Ornithine L-Aspartate (LOLA)**
- **IV hydration**
- **Dietary protein restriction**
- **Zinc supplementation**
- **Renal replacement therapy (severe cases)**

In cases with confirmed S. maltophilia bacteraemia, TMP-SMX must be initiated in addition to management of the primary pathology. The co-existence of bacteraemia and CAUTI creates a dual source of potential ammonia load and requires parallel antibiotic strategies targeted at both organisms, supported by susceptibility data.

6. Conclusion

This case of acute hyperammonaemia encephalopathy — 131.8 mcg/dL with entirely normal liver function, normal fibro scan, and no clinical stigmata of chronic liver disease — **underscores the imperative that clinicians think beyond hepatic aetiology** when faced with an encephalopathic patient. CAUTI-associated hyperammonaemia, mediated by urease-producing organisms (ESBL E. coli in this case), is a well-established but underrecognized mechanism.

The novel dimension introduced by this case is the concurrent isolation of Stenotrophomonas maltophilia from blood cultures — a finding without precedent in the published hyperammonaemia literature. The biochemical properties of S. maltophilia, including its **serine/metalloprotease arsenal (StmPr1-3), genome-encoded nitrogen assimilation machinery, active deaminase activity, and characteristic ammonia generation in culture, provide a plausible mechanistic framework for its role in systemic ammonia dysregulation in the bacteraemia state.**

Formal in vitro studies of S. maltophilia ammonia generation in blood-equivalent media at physiological temperature, and a case registry of S. maltophilia bacteraemia-associated hyperammonaemia, are urgently needed.

TAKE-HOME MESSAGES FOR ALL SPECIALTIES

- Hyperammonaemia without liver disease = think CAUTI and infection first. Always check serum ammonia in unexplained encephalopathy.
- Concurrent S. maltophilia bacteraemia in a hyperammonaemia patient is not an incidental finding — treat it, investigate it.
- TMP-SMX is the drug of choice for S. maltophilia — do not rely on carbapenems or broad-spectrum beta-lactams.
- The bacteraemia state — not urinary colonisation — provides maximal systemic substrate for proteolytic ammonia generation.
- This case is an index observation — report similar cases to build the evidence base.

Functional outcome of Tardy ulnar nerve palsy treated by subcutaneous transposition of ulnar nerve: A case report highlights:



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Tardy ulnar nerve palsy is a chronic clinical condition characterized by delayed onset neuropathy.

The most common cause of symptoms is malunion of lateral condyle of humerus, which leads to a cubitus Valgus that stretch the ulnar nerve.

Minimal Valgus deformity could still manifests as Tardy ulnar nerve palsy.

Introduction:

Tardy Ulnar nerve palsy is a chronic clinical condition characterized by delayed onset ulnar neuropathy. Ulnar nerve is vulnerable because of its curvature around elbow joint.



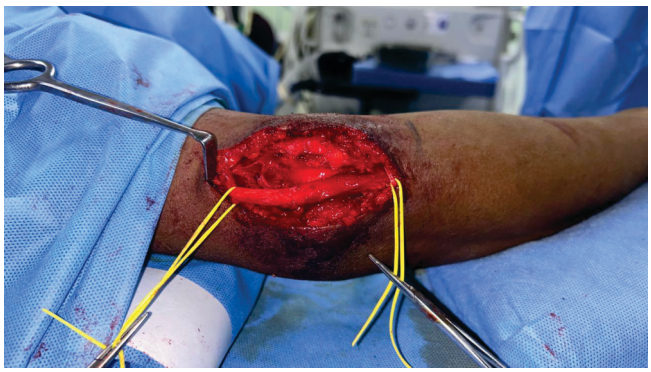
Any pathological condition which alters normal anatomy of elbow joint can lead to stretching or irritation of nerve, late onset neuropathy was deemed to results from fractures of lateral condyle of humerus with malunion leading to a cubitus Vulgas deformity. Tardy ulnar nerve palsy can also result from cubitus varus deformity.

The recovery of ulnar nerve will depend on the severity of nerve injury, preoperatively. Most authors, recommend anterior transposition of Ulnar nerve to release the compression.

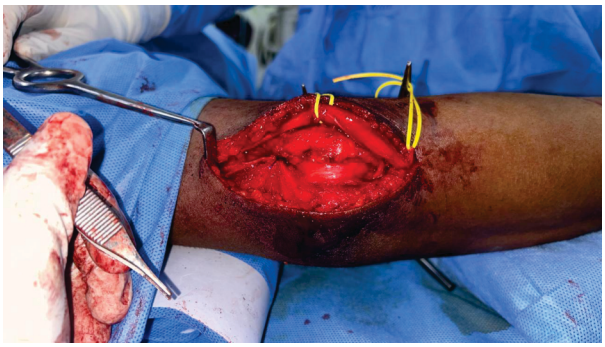
Case presentation:

A 40 years old male was referred with complaints of clawing of left ring and little finger for six months. Patient also complaints of numbness and tingling sensation on ulnar side of forearm left ring finger and little finger. The patient was treated with subcutaneous anterior transposition of Ulnar nerve with ulnar









nerve neurolysis. The ulnar nerve is transposed anterior to medial epicondyle, thereby eliminating longitudinal, traction forces applied to the nerve during elbow flexion.

Postoperatively, patient received Transcutaneous Electrical Nerve Stimulation (TENS), and structured



physiotherapy with Knuckle Bender splint. Patient recovered well significantly postoperatively.

Discussion:

The ulnar nerve is relatively fixed, behind medial epicondyle of humerous. Any increase in a valgus deformity at elbow, would lead to stretching of the nerve resulting in neurapraxia. This condition is temporary and usually recovers with remyelination of Axons that takes about. There was a history at trauma to left elbow when he was 10 years old, which was Managed conservatively.

Physical examination revealed clawing at left ring and little finger, wasting a hypothenar muscle and dorsal interosseous muscles and weakness of intrinsic muscles of fingers, loss of adduction of little finger. There is a slight Valgus deformity observed on left elbow with carrying angle of MRI revealed old non-United mildly displaced and comminuted Fracture of lateral Condyle of humerus. With thickened ulnar



nerve and loss of normal fascicular pattern of nerve.

Nerve conduction study revealed features of severe degree of left ulnar neuropathy with entrapment at level of elbow.

EMG showed re-innervation potential of flexor carpi ulnaris, Adductor digiti Mini and dorsal interosis three weeks to three months.

If deformity force is progressive, it might injure the axon within the endoneural tube of the nerve with intraneural, scarring and fibrosis. Tardy Ulnar nerve palsy has a variety of etiological factors, including old fractures around elbow, arthritis, congenital anomalies, etc, flowever old supracondylar or lateral condylar fracture remains common cause.

This patient was treated with ulnar nerve transposition without osteotomy or correcting the non-union of lateral condo because there is no significant deformity.

Conclusion:

The treatment of choice for Tardy Ulnar nerve palsy is anterior subcutaneous ulnar nerve transposition with Ulnar nerve neuropathy, which shows excellent post op recovery.



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